of anal HPV infection among populations at high risk, particularly MSM (*1278*,*1289*,*1290*). No standard HPV-based algorithms exist for anal cancer screening due to the high prevalence of high-risk HPV infection among groups at risk.

Treatment of Anal High-Grade Squamous Intraepithelial Lesion

Multiple office-based treatments exist for anal HSIL, including ablative methods (e.g., laser, electrocautery, or infrared coagulation) and topical patient-applied therapies (e.g., imiquimod). Recurrence rates with both provider-applied and patient-applied treatments are high, ranging from approximately 50% at 1 year to 77% after 3 years (*1289*,*1292*,*1293*). In addition, evidence exists that HSIL might spontaneously regress without treatment (*1294*,*1295*). Shared decision-making about treatment for anal HSIL is recommended because of limited data on the natural history of anal HSIL, including factors related to progression or regression of lesions.

Viral Hepatitis

Hepatitis A Virus Infection

HAV infection has an incubation period of approximately 28 days (range: 15–50 days) (*1296*). HAV replicates in the liver and is shed in high concentrations in feces from 2–3 weeks before to 1 week after the onset of clinical illness. HAV infection produces a self-limited disease that does not result in chronic infection or chronic liver disease. However, approximately 10% of patients experience a relapse of symptoms during the 6 months after acute illness. Acute liver failure from hepatitis A is rare (overall case-fatality rate: 0.5%). The risk for symptomatic infection is directly related to age, with approximately 70% of adults having symptoms compatible with acute viral hepatitis and the majority of children having either asymptomatic or unrecognized infection. Antibody produced in response to HAV infection persists for life and confers protection against reinfection (*1297*).

HAV infection is primarily transmitted by the fecal-oral route, by either person-to-person contact or through consumption of contaminated food or water (*1298*). Transmission of HAV during sexual activity probably results from fecal-oral contact. Although viremia occurs early during infection and can persist for weeks after symptom onset, bloodborne transmission of HAV is uncommon (*1299*). Transmission by saliva has not been demonstrated.

In the United States, of the hepatitis A cases accompanied by risk information, a particular risk was identified among only 23.8% (*13*,*372*). Among cases with a risk factor identified, a recognized foodborne or waterborne outbreak was the most commonly identified risk (49.6%). Other infection sources identified in the United States include MSM; persons who use injecting drugs; sexual and household contacts; those experiencing homelessness; international travelers; those with children attending a nursery, childcare, or preschool; and persons working in such settings (*13*,*372*).

Diagnostic Considerations

Diagnosis of HAV infection cannot be made on a clinical basis alone but requires serologic testing. Presence of IgM antibody to HAV is diagnostic of acute HAV infection. A positive test for total anti-HAV indicates immunity to HAV infection but does not differentiate current from previous HAV infection. Although usually not sensitive enough to detect the low level of protective antibody after vaccination, anti-HAV tests also might be positive after hepatitis A vaccination.

Treatment

Patients with acute HAV infection usually require only supportive care, with no restrictions in diet or activity. Hospitalization might be necessary for patients who become dehydrated because of nausea and vomiting and is crucial for patients with signs or symptoms of acute liver failure. Medications that might cause liver damage or are metabolized by the liver should be used with caution among persons with HAV infection.

Prevention

Vaccination is the most effective means of preventing HAV transmission among persons at risk for infection (e.g., MSM, injecting drug users, and persons with chronic liver disease) who did not receive hepatitis A vaccination during childhood. Hepatitis A vaccines are prepared from formalin-inactivated, cell-culture–derived HAV. Two monovalent vaccines (Havrix and Vaqta are approved by FDA for persons aged ≥12 months (Table 3). These vaccines are available for eligible children and adolescents aged <19 years through the VFC program (https://www.cdc.gov/vaccines/programs/vfc/index.html). Administered IM in a 2-dose series at 0 and 6–12 months, hepatitis A vaccines induce protective antibody levels among virtually all adults. By 1 month after the first dose, 94%–100% of adults have protective antibody levels, and after a second dose, 100% achieve protective levels (*1297*,*1300*,*1301*). Kinetic models of antibody decrease among adults indicate that protective levels persist for >40 years (*1302–1304*). A study of Alaska Natives demonstrated that seropositivity for hepatitis A persists for >20 years after completing 2-dose vaccination at age 12–21 months (*1302*). Anti-HAV persistence of >20 years was demonstrated among immunocompetent adults vaccinated